simultaneous motor discharge from a nerve centre to all the muscular fibres; the contraction is, on the other hand, a progressive process passing over the auricular walls in a wave-like fashion.

9. A reversal of the normal sequence of the heart's contraction can be induced and kept up for a considerable time by applying to the ventricles a series of single stimulations (*e.g.*, induction shocks) at a rate somewhat more rapid than that of the spontaneous rhythm of the organ.

V. "Inhibition of the Mammalian Heart." By JOHN A. McWILLIAM, M.D., Professor of the Institutes of Medicine in the University of Aberdeen. Communicated by Professor M. FOSTER, Sec. R.S. Received May 3, 1888.

The following conclusions are based upon a long series of experiments performed upon the cat, dog, rabbit, rat, hedgehog, and guinea-pig, the cat being the animal commonly used. The animals were anæsthetized, usually with chloroform; artificial respiration was kept up; the thorax and often the pericardial sac were laid open, and the action of the heart was examined with the aid of the graphic method.

Section of the Vagi.

The results of section of both vagi vary according to the conditions obtaining at the time the nerves are cut—according to the amount of controlling influence exercised by the medullary cardio-inhibitory centre upon the heart. When the cardio-inhibitory centre is inactive, section of the vagi causes no appreciable change in the heart's action. On the other hand, section of the nerves at a time when the controlling influence of the medullary centre is acting to a decided extent, is followed by very pronounced results—by an increase not only in the rate of the cardiac beat, but also in the contraction force of both the auricles and the ventricles. There is a marked augmentation in the strength of the beats; the change in the energy of the auricular contractions is usually *more* extensive than that occurring in the case of the ventricles.

Stimulation of the Vagus Nerve.

The latent period of vagus stimulation varies remarkably in different conditions; there is often a period of many seconds before the heart stands still.

When the vagus nerve is stimulated so as to *slow* the heart, it is usually seen that the inhibitory influence is not of maximal intensity at its first manifestation, but goes on increasing for some time.

Effects of Vagus Stimulation on the Auricles.

1. The vagus appears as a rule to influence the auricles more readily and more powerfully than the ventricles.

2. Vagus stimulation leads to a slowing or an arrest of the rhythmic beat, and a very marked weakening of the contraction force.

The recommencing auricular beats that occur when the period of inhibition is passing away are very weak; and any contractions excited by direct stimulation (*e.g.*, with induction shocks) during the period of standstill are strikingly enfeebled.

3. Vagus stimulation causes a pronounced depression of the excitability of the auricular tissue to direct stimulation.

During the period of inhibition resulting from vagus stimulation it is much more difficult than usual to excite an auricular beat by direct excitation; a much stronger stimulus is necessary to elicit any contraction at all.

4. The tone of the auricular muscle appears to be markedly diminished.

5. These results occur when the vagus is stimulated, even when the superior and inferior venæ cavæ have been clamped, so that the cavities of the heart are no longer filled with blood.

6. The vagus nerve seems to exert a powerful influence of a more or less direct nature on the muscle itself, not merely by inhibiting or weakening the motor impulses which are commonly assumed to pass from nerve centres in the heart to the muscular fibres. For if it were true that the vagus acted simply by depressing the motor centres of the heart, it is very difficult to conceive how the responsiveness of the auricular muscle to direct stimuli should be so greatly diminished, and how the contraction force should be so strikingly reduced when the auricular muscle is made to contract by induction shocks applied to the auricular tissue.

It would seem that whatever changes the vagus may induce in the nerve-cells and ganglia occurring plentifully in the auricles, it can also exert an important influence on the contractile tissue itself.

7. Upon the whole, the influence of the vagus nerve upon the mammalian auricles presents a close parallelism to what holds good in the auricles of many cold-blooded animals.

Effects of Vagus Stimulation on the Ventricles.

Besides causing slowness or standstill, the vagus can cause other important changes in the ventricular part of the heart.

1. The contraction force is markedly diminished. When a period of standstill has ended, the recommencing beats are usually weak; and beats excited by direct stimulation (*e.g.*, single induction shocks) during the period of standstill are of diminished size.

When vagus stimulation does not cause complete standstill, but only a marked slowing, the strength of the slow ventricular beats is usually much less than the normal.

The reduction in contraction force does not bear any *constant* relation to the degree of slowing. While *all* the slow beats are weakened *in some degree*, a beat occurring after a long pause is *sometimes* decidedly *stronger* than one occurring after a shorter pause; on the other hand, the converse more often holds good—a beat occurring after a long pause is *weaker* than a beat occurring after a shorter pause.

The depression of contraction force does not appear to depend on over-distension of the ventricles during the slowing or standstill; nor upon the fall of arterial pressure that occurs and involves a diminished resistance to the ventricular systole and a change in the coronary circulation.

The force-depressing effects of vagus stimulation can still be seen (1) when the superior and inferior venæ cavæ have been clamped; or (2) when the pulmonary artery or (3) the aorta has been clamped; or (4) when *all* these vessels have been clamped before the vagus stimulation.

2. When slowing or arrest of the ventricular action occurs as a result of vagus stimulation, there is a marked change in the shape and duration of the ventricular curves; the degree of change stands in close relation to the length of the pause preceding each beat. The curves become broader near the top, and their duration is increased. The longer the interval preceding a curve the broader the curve is, and the more markedly is it prolonged. These features are not abolished when the superior and inferior venæ cavæ have been clamped before the vagus stimulation; nor when the aorta or the pulmonary artery, or all these vessels, have been clamped.

3. The vagus appears to inhibit the spontaneous rhythmic tendency inherent in the ventricles; the ventricular standstill does not appear to be due simply to the standstill of the rest of the heart.

4. At the same time the absence of auricular beats of any considerable strength is *usually* a necessary condition for the occurrence of a protracted ventricular standstill. It commonly but not invariably happens that if the auricles are artificially excited to contract during the period of cardiac standstill, the ventricles beat also in sequence to the artificially excited auricular contraction.

5. When the heart begins to beat after a period of inhibition, the order of contraction most commonly seen is that which obtains normally—ostial parts of the great veins; auricles; ventricles. But sometimes the ventricles recommence, and give one or more beats before any contraction occurs in the other parts of the heart.

6. There are sometimes seen evidences of the occurrence under vagus influence of a block in the propagation of the contraction from

auricles to ventricles. At certain phases of vagus stimulation the ventricles often fail to respond to auricular beats, while at the same time there is evidence to show that this is due not to a depression of the ventricular excitability, but to a break in the transmission of the contraction from the auricles.

7. The *maximum* intensity of the inhibitory influence exerted by vagus stimulation often obtains at the same time in the auricles and the ventricles. But frequently the auricles become greatly depressed, while the ventricular beats are of undiminished size, or are only beginning to be affected; in rare cases the ventricular contraction force becomes reduced more suddenly than the auricular.

8. The effects of vagus stimulation on the ventricles may be in some measure counteracted by the application to the ventricular surface of a series of stimulations (*e.g.*, single induction shocks) at about the normal rate of the heart's action. An artificially excited series of beats is thus caused; these beats give curves of approximately normal form and duration, and they are much stronger than any slowly occurring spontaneous beats that appear after the standstill has lasted for some time; they are also much stronger than single beats excited (by induction shocks) at long intervals during the standstill. The beats of the artificially excited series (at normal rate) are still decidedly weaker than normal beats.

On the Existence of a Local "Inhibitory Area" in the Heart.

By stimulation of a certain locality on the dorsal aspect of the auricular surface, certain striking effects are obtained. In the cat and dog the area in question is elongated in shape, and is situated over the inter-auricular septum, its long axis running parallel with the plane of the septum. It extends downwards to within a short distance of the coronary sinus. At the right side of the area lies the termination of the vena cava inferior.

Many nerves course downwards through this region; there are also numerous nerve-cells and ganglia. These, however, are not confined to the area in question, but occur in considerable number over the dorsal aspect of the left ventricle, especially in its septal half. The nerves appear to be derived to a considerable extent from the left vagus. The majority of the fibres are non-medullated, but medullated fibres are also present (cat). Ganglia occur in special abundance near the auriculo-ventricular groove.

Stimulation of this area with an interrupted current gives results that stand out in sharp contrast to those obtained by stimulating other parts of the auricular wall, *e.g.*, the appendix. Stimulation of the latter causes an acceleration of both auricles and ventricles. The auricles contract with great rapidity, so that they present a peculiar

fluttering appearance; the ventricles beat much more rapidly than before, though they do not keep pace with the auricles.

On the other hand, stimulation of the inhibitory area, while it causes a rapid fluttering action of the auricles, induces either a very marked slowing, or a complete standstill in the ventricles. This result is a mixed one—ventricular inhibition, resulting from stimulation of certain structures in the inhibitory area, and auricular acceleration, in all probability due to an escape of the stimulating current to the excitable auricular tissue.

The inhibitory effects on the ventricle much resemble those caused by vagus stimulation. There is depression of the ventricular contraction force, and changes in the shape and duration of the ventricular curves similar to those occurring under vagus influence. Stimulation of the inhibitory area and of the vagus are both rendered ineffective by the administration of atropine.

But there are certain points of difference :—

(1.) The strength of current necessary to inhibit the ventricles is very much less when the current is applied to the inhibitory area than when it is applied to the vagus.

(2.) Stimulation of the inhibitory area remains effective in arresting the ventricular action, after curare has been administered in such amount as to cause stimulation of one or both vagi in the neck to be entirely without inhibitory result.

(3.) In many instances when the vagi have become exhausted, or have lost their inhibitory power from less definite causes, the inhibitory area remains effective.

It seems clear from the very different relation borne by the inhibitory area to certain poisons, to the strength of stimulating current necessary, to exhaustion, &c., that in exciting this area we are dealing with structures of a more or less special nature, differing markedly in their character from the ordinary inhibitory fibres running in the trunks of the vagus nerves.

The important structures of the inhibitory area are situated superficially; they may be readily paralysed by the application of a few drops of a 4 per cent. solution of cocaine hydrochlorate, or of strong ammonia.

The region in question does not contain a motor centre for the heart muscle. Destruction of this area does not arrest the spontaneous rhythm of the organ (which indeed originates in parts some distance removed from the inhibitory area, viz., in the ostial parts of the great veins, especially the vena cava superior and the pulmonary veins). Nor is the propagation of the contraction from one part of the heart to another in any way deranged or interfered with.

The inhibitory area probably contains structures to which many at

least of the inhibitory fibres of the vagus go, there to come into intimate relation with the cardiac mechanism.

Effect of Stimulation of Ostial Parts of Great Veins in certain Abnormal Conditions.

At certain stages of the process of asphyxia, and in the dying heart, there is often seen a very remarkable alteration in the behaviour of the ostial parts of the great veins towards direct stimulation with interrupted currents. In such circumstances, an inhibition of the spontaneous rhythmic action of these parts may often be seen as a result of direct stimulation, whereas in the normal state such a stimulation is productive of immediate and striking acceleration.

VI. "On the Structure of the Electric Organ of *Raia circularis*." By J. C. Ewart, M.D., Regius Professor of Natural History, University of Edinburgh. Communicated by Professor J. Burdon Sanderson, F.R.S. Received April 30, 1888.

(Abstract.)

This paper gives an account of the structure of the cup-shaped bodies, which, as mentioned in a previous paper read 26th April, 1888, make up the electric organs of certain members of the skate family. The structure of these electric cups has been already studied in three species of skate, viz.: *Raia fullonia*, *R. radiata*, and *R. circularis.* The present paper only deals with the electric organ of *R. circularis.* It shows that the cups in this species are large, well-defined bodies, each resembling somewhat the cup of the familiar "cup and ball." The cup proper, like the disks of *R. batis*, consists of three distinct layers, (1) the lining, which is almost identical with the electric plate of *R. batis*; (2) a thick median striated layer; and (3) an outer or cortical layer. The lining or electric plate is inseparably connected with the terminal branches of the numerous nerve-fibres, which, entering by the wide mouth in front, all but fill the entire cavity of the cup, and ramify over its inner surface, the intervening spaces being occupied by gelatinous tissue. This electric layer, which is richly nucleated, presents nearly as large a surface for the terminations of the electric nerves as the electric plate which covers the disk in *R. batis* and *R. clavata.* The striated layer, as in *R. batis*, consists of numerous lamellæ, which have an extremely contorted appearance, but it differs from the corresponding layer in *R. batis*, in retaining a few corpuscles. The cortical layer very decidedly differs in appearance from the alveolar layer in *R. batis.* It is of considerable thickness, contains large nuclei,